Second Edition

Handbook of FORENSIC PATHOLOGY

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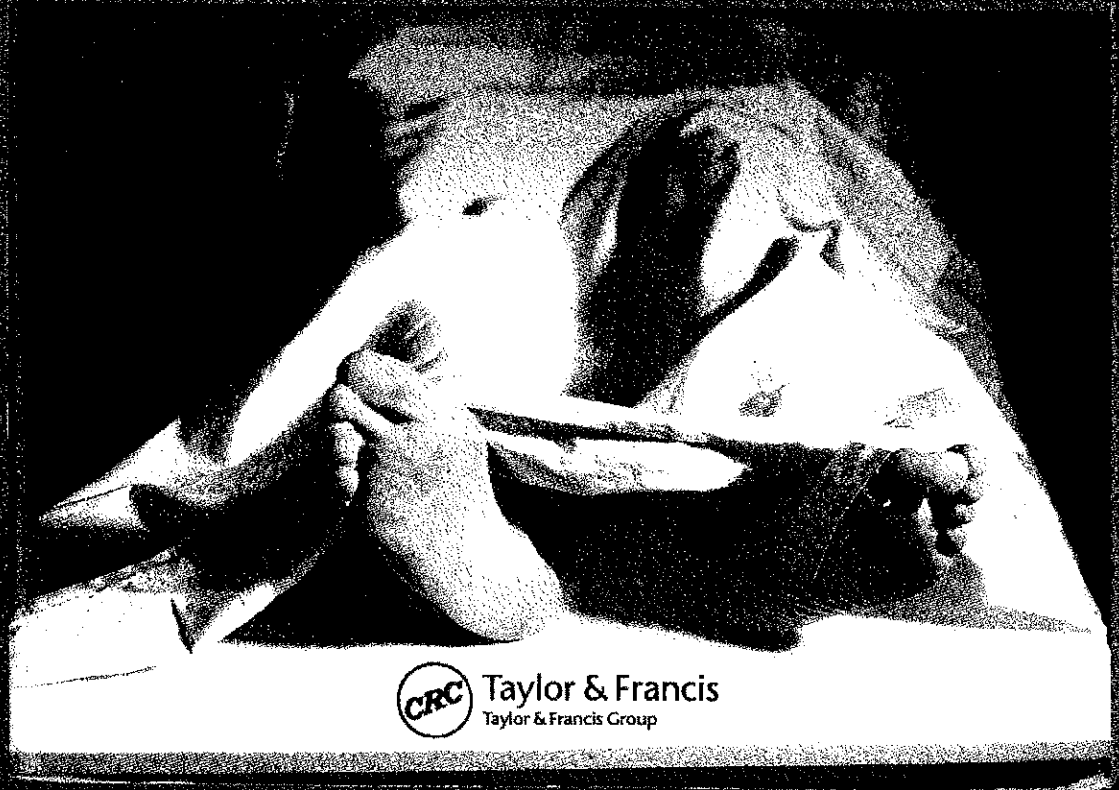

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levels of these catecholamines did not occur during the struggle but in the 3 minutes immediately following cessation of the exercise. Thus, the individual is in a hyperadrenergic state due to very high levels of NE and E.

4. Young et al.[2] found that following cessation of exercise, the potassium level fell rapidly, returning to approximately normal levels in 5 minutes. The maximum rate of fall occurred within **the first or second minute post-exercise**.
5. Low blood potassium predisposes to prolongation of the QT interval, development of *torsade de pointes* (uncommon variant of ventricular tachycardia), and sudden cardiac death.
6. Thus, the physiological effects of strenuous exercise are increasing levels of NE and E peaking in the minutes after cessation of a struggle and rapidly dropping levels of potassium in the minutes after cessation of a struggle with both effects predisposing to sudden death.

C. Other factors:
1. Cocaine stimulates the heart indirectly by activating the sympathetic nervous system and directly at the synapses. Cocaine blocks the reuptake of NE at the synapses causing its accumulation. This results in hyperstimulation of the heart and coronary arteries.
2. There is evidence of polymorphism of α and β heart receptors that results in increased quantities of NE in the synapses and increased sensitivity to the NE by the heart receptors.
3. Individuals with intrinsic mental disease such as schizophrenia and bipolar disease are predisposed to develop excited delirium. Their medications may be cardiotoxic.
4. Other drugs such as diphenhydramine may cause excited delirium.

D. Death due to EDS is due to a cardiac arrhythmia secondary to a hyperadrenergic state and a decrease in potassium due to the synergistic, physiological effects of:
1. Excited delirium
2. The struggle
3. Drugs, illegal and therapeutic
4. Mental disease
5. Possibly a genetic predisposition

III. HOGTYING — POSITIONAL/RESTRAINT ASPHYXIA

A. In hogtying, an individual is placed in a prone position, their hands are tied or cuffed together behind their back, and their ankles are bound and tied to their wrists. Based on experiments

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in the 1980s, it was concluded that if you hogtie an individual, you interfere with their ability to breathe and, thus, can cause death. These deaths were then said to be due to positional/restraint asphyxia.

B. Police stopped use of the hogtie, but individuals kept dying.

C. By this time, the idea of these deaths being due to positional asphyxia was embedded in society and the concept was retained, though not the mechanism. It was claimed these deaths were due to the officers or medical personnel lying on the individual or applying pressure on the thorax with knees.

D. Research by Chan et al.[3] determined that the original experiments were in error. He found that while placing an individual facedown in the hogtie position following strenuous exercise, e.g., a struggle, did produce restrictive pulmonary functioning, as measured by pulmonary function test, these results were not clinically relevant. There was no evidence of hypoxia.

E. Subsequent tests in which weights were applied to the thorax also did not produce clinically relevant decreases in pulmonary functioning. Thus, there is no proof that ordinary force placed on an individual by kneeling on them or lying across their body compromises respiration.

IV. CHOKE HOLD–RELATED DEATHS

Deaths have been reported as being due to the use of **choke holds**.[4] In such deaths, there are two possible mechanisms for death:

A. First is compression of the carotid arteries with resulting cerebral hypoxia. But in such cases:

1. Compression of the neck would have to be for at least 2 to 3 minutes to cause cessation of respiration.
2. Such an individual would be expected to respond to cardiopulmonary resuscitation (CPR) but this is not the situation in the cases reported.
3. One would expect petechiae of at least the conjunctivae and sclerae since such a case is essentially a strangulation. Again, this is not usually seen.
4. Thus, there is no evidence that such deaths are due to strangulation.

B. The other proposed mechanism of death is cardiac arrest due to severe bradycardia secondary to stimulation of the vagus nerve or carotid receptors. The problem with this explanation is twofold:

1. A search of the literature for well-documented deaths due to vasovagal stimulation reveals them to occur almost exclusively in elderly individuals with underlying severe vascular disease.[5]